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L3   ANSWER 1 OF 2     MEDLINE on STN                    DUPLICATE 1
AN   2003352190     MEDLINE
DN   PubMed ID: 12884866
TI   Caspase-8 and caspase-10 activate NF-kappaB through RIP, NIK and IKKalpha
     kinases.
AU   Shikama Yoshiaki; Yamada Masao; Miyashita Toshiyuki
CS   Department of Genetics, National Research Institute for Child Health and
     Development, 3-35-31 Taishido, Setagaya-ku, Tokyo 154-8567, Japan.
SO   European journal of immunology, (2003 Jul) 33 (7) 1998-2006.
     Journal code: 1273201. ISSN: 0014-2980.
CY   Germany: Germany, Federal Republic of
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals
EM   200308
ED   Entered STN: 20030730
     Last Updated on STN: 20030828
     Entered Medline: 20030827
AB   NF-kappaB regulates the expression of various genes involved in cell
     growth and differentiation, immune response and inhibition of apoptosis.
     Recently, some death effector domain (DED)-containing proteins, such as
     FADD and c-FLIP were reported to activate NF-kappaB.  We previously
     reported that the prodomain-only isoforms of caspase-8 and -10
     (PDCasp8/10), containing two DED motifs, could inhibit Fas-mediated
     apoptosis.  Here, we demonstrate that these isoforms also activate
     NF-kappaB, implying this to be one of the mechanisms by which these
     polypeptides inhibit apoptosis.  The GST pull-down assay revealed that,
     among upstream kinases that activate NF-kappaB, only NIK and RIP, but not
     RICK or IKKalpha/beta, could directly bind to PDCasp8/10.  In addition,
     both modules ofDED in PDCasp8/10 were required for these interactions as
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well as NF-kappaB activation. Experiments using a kinase-dead mutant of IKKalpha and an **RIP** mutant lacking a **kinase** domain, both of which function as **dominant-negative** mutants for their wild-type counterparts, blocked PDCasp8/10-mediated NF-kappaB activation. Using small interfering RNA technology, we further demonstrate that the down-regulation of IKKalpha but not IKKbeta significantly inhibits PDCasp8-mediated NF-kappaB activation. Taken together, these results suggest that caspase-8 and -10 have roles in a non- or anti-apoptotic signaling pathway leading to NF-kappaB activation through RIP, NIK and IKKalpha.

L3 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2002:616256 CAPLUS
DN 137:181594
TI Dominant-negative variants of human protein kinases that inhibit the phosphorylation activity of their active enzyme isoforms
IN Levine, Zurit; Bernstein, Jeanne
PA Compugen Ltd., Israel
SO U.S. Pat. Appl. Publ., 170 pp., Cont.-in-part of U.S. Ser. No. 724,676.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2002110811	A1	20020815	US 2001-771161	20010126
PRAI	IL 2000-135619	A	20000512		
	IL 2000-136776	A	20000615		
	US 2000-724676	A2	20001128		

AB The present invention concerns 91 nucleic acid sequences and amino acid sequences of variants of various human kinases, i.e. of sequences which inhibit activity of kinases in a dominant manner. The variants lack a domain or region required for phosphorylation, and thus may be dominant-neg. kinases obtained by alternative splicing of known original sequences of the kinase genes. The novel dominant-neg. kinase variants of the invention are not merely artificially truncated forms, fragments or mutations of known genes, but rather novel sequences which naturally occur within the body of individuals. The invention also concerns pharmaceutical compns. and detection methods using these sequences.

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